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Research Interests

Our research focuses on the molecular mechanisms of renal fibrosis, particularly on those changes occurring during the inflammatory and fibrotic stages.
To study renal interstitial fibrosis, we use the unilateral ureter obstruction (UUO) and protein overload models, and for diabetic nephropathy, we use the streptozotocin (Stz) and db-/- models. We established that Timp-1 deficiency does not alter the degree of interstitial fibrosis in the murine protein overload or UUO models, possibly due to a genetic redundancy with other genes, such as Timp-2. We established the important fibrogenic role of PAI-1, proving its importance as a fibrosis-promoting gene. Similar results were observed in two diabetic nephropathy models (Stz and db-/-) in combination PAI-1+/+ and
PAI-1-/- mice. We reported that the uPAR gene attenuates the renal fibrosis, possibly mediated by a urokinase-dependent, yet plasminogen-independent, system. We demonstrated the importance of the IL-6 family of cytokines during the renal inflammatory process prior to the chronic fibrotic stage. The results indicated a protective role of gp130-diminishing fibrosis. It is possible that a "talking" system exists between the uPAR and the IL-6 family of cytokines in the regulation and/or progression of fibrosis.

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